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THE PROBLEM OF HEART DISEASE AS IT STANDS TODAY*

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The magnitude and the seriousness of the problem of heart disease is very graphically brought to us by this single fact: in 1935, 312,333 persons died in the United States of heart disease. The challenge of this very real problem is not going unanswered: the resources of the medical profession are being applied to its solution as they rarely before have been used. Furthermore, this intensive effort is bearing fruit; for we have made tremendous strides in our knowledge of heart disease in the last twenty years. But in spite of the progress that has been made, there are still before us many fundamental and major problems that must be solved ultimately if we are ever to approach our goal, which, as expressed by the American Heart Association, is the prevention and relief of heart disease. When we are in the midst of any heated campaign, it is perhaps wise to stop occasionally and take stock: to put down on the one hand our gain and progress, and against this consider our failures and the obstacles yet to be overcome. I should like to do this with you today.

THE DIAGNOSIS OF HEART DISEASE

We have made our greatest progress perhaps in the field of diagnosis. Indeed, this field of cardiology (and it is a very important one) can be looked upon as almost a finished chapter. It seems to me that the eliciting of evidence and its interpretation into a diagnosis is approaching its fullest development. If profress in cardiology is slow, it is certainly not because of lack of diagnostic knowledge.

One factor that has greatly broadened our diagnostic ability is the perfection of the electrocardiographic method. Let me remind you that as recently as 1912, coronary occlusion was thought to be incompatible with life; this diagnosis was never made upon a living person. Now all of us confidently make this diagnosis without ever seeing the patient. We can look at an electrocardiogram and say with at least ninety-five per cent of certainty not only that a coronary occlusion has or has not occured, but we can say which portion of the heart muscle has been involved, and we can give a fairly accurate estimate of the amount of muscle infarcted. Ninety-five per cent is certainly approaching perfection and is as accurate as we have any right to expect any method in medicine to be.

The x-ray is another graphic method that has tremendously extended our diagnostic accuracy. In practically every instance of cardiac disease, there is enlargement of some chamber of the heart. Whether enlargement exists or not is therefore the most important factor that we wish to discover. While this can be determined often without x-ray help, many times it cannot. Because it so definitely determines the heart size and because this is so important to know, Sir Thomas Lewis once stated that if he could have but one method of examining the heart, he would without question select the x-ray.

Still another great improvement in the diagnosis of heart disease has come about through a better understanding of the significance of murmurs. A diastolic murmur is probably always an evidence of organic disease; systolic murmurs frequently are without significance; they may be present in perfectly sound hearts. Not only this: we have learned that even when murmurs do result from cardiac disease, they are not always the

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most accurate finding upon which to base an estimate of the severity of the damage: there are other factors, such as the degree of cardiac enlargement, etc., that much more accurately determine the prognosis. Let us take as an example, mitral stenosis. If one bases a prognosis on the loudness or character of the murmur, one will go astray: the factor upon which one can best base a prognosis is the degree of strain put upon the right ventricle as determined by the intensity of the second pulmonic sound, and the size and shape of certain portions of the heart.

THE PHYSIOLOGY OF HEART DISEASE

Among the various phases of cardiology in which we have made conspicuous progress, physiology is second only to diagnosis. I should like to discuss briefly a few phases of this subject.

The Arrhythmias. Not so many years ago, the cardiac arrhythmias were most puzzling. The various irregularities of the heart were not differentiated, and they were all thought to be a manifestation of a serious cardiac disorder. Chiefly through the work of Sir James MacKenzie and Sir Thomas Lewis, we now understand the mechanism of practically all the arrhythmias. This clarifying of the physiology of the arrhythmias is a beautiful example of the value that pure scientific research may have for actual clinical medicine: for only after the physiology of the arrhythmias became accurately known, were we in a position to study and treat them intelligently. Since we now know what fundamental property of the heart muscle is at fault in the various arrhythmias, we can logically decide whether digitalis or quinidine, or some other drug or procedure is indicated. We cannot say this for all phases of our treatment of heart disease, but we can justifiably say that our treatment of the cardiac arrhythmias is on a firm scientific basis.

Heart Failure. There is much that we still must learn concerning the physiology of heart failure. When one is faced with any baffling problem, the logical procedure to follow is to reduce the problem to its component parts and then attempt to solve each part separately.

We have simplified the problem of heart failure considerably by learning that there are several types of heart failure. Not every function of the heart need fail, nor is it necessary for every chamber to fail. We, therefore, can divide heart failure into several subdivisions. The following will suffice for our discussion today: (1) left ventricular failure; (2) right ventricular failure; (3) failure of diastolic filling; (4) anginal failure.

Left Ventricular Failure. When the left ventricle loses its ability to correctly do its work which is to pump blood through the arterial system, the resistance in the left auricle and throughout the whole venous side of the pulmonary bed rises, and pulmonary congestion results. The symptoms of this are breathlessness and cyanosis. In an advanced form, it appears spontaneously in paroxysms in the form that we speak of as paroxysmal dyspnoea or cardiac asthma. If the left ventricular failure goes beyond this, pulmonary edema develops.

These dramatic events develop only when the left ventricle is severely taxed and nearly defeated. Early left ventricular failure also has its signs. Breathlessness on exertion is one. Another is congestion of the lungs. Before rales or other clinical signs are demonstrable, congestion will exist, which can be detected by the x-ray or the fluoroscope. The demonstration of such cryptic congestion may be very helpful.

When left ventricular failure shows itself as asthmatic attacks, it may be extremely difficult to know whether the asthma is bronchial or cardiac. A helpful test is the determination of the circulation time. In cardiac asthma this will be prolonged: in bronchial asthma, it will not, provided there is no complicating heart disease present. There are a number of methods of determining the circulation time that are simple, easily applied, and accurate.

We see pure left ventricular failure very frequently without any evidence of right ventricular failure. It is perhaps most common in hypertension, where the strain of overcoming the greatly increased peripheral resistance becomes temporarily too great for the resources of the left ventricle. We also see it quite frequently in acute coronary occlusion. Its development is always a threat in the early stages of such an accident.

Right Ventricular Failure. When the right ventricle fails, the blood is pushed back into the right auricle. This dams the blood back, so to speak, into the venous system. The clinical sign of right ventricular failure is therefore an increased venous pressure and the changes which result from this, such as congestion of the liver, edema, etc.

While one frequently sees left ventricular failure alone, it is quite uncommon to encounter right ventricular failure without accompanying failure of the left heart. One reason which has only recently been appreciated is the fact that the commonest cause of right ventricular failure is preceding left-sided failure (1). For when left heart failure produces a greatly increased resistance in the venous side of the pulmonary circulation, the work of the right ventricle is greatly increased and its defeat follows.

Failure of Diastolic Filling (2). This variety of heart failure frequently possesses the properties of both left and right ventricular failure. However, the mechanism of its production is so different that it deserves a separate classification. In ordinary right and left ventricular failure, the fault lies in the failure of the heart's propulsive force: it lacks the power to properly eject blood. In the failure of diastolic filling, the end result is the same: an insufficient amount of blood is ejected from the ventricles into the peripheral circulation. The reason for this failure is very different, however: the ventricles fail to put out sufficient blood during systole simply because they do not become properly filled during diastole.

We see this type of failure generally under two circumstances. The first is when the heart beats very rapidly and there simply is not time during the very short diastole for sufficient blood to enter the ventricles from the auricles. The second is seen when there exists a dense adherent pericardium. Such a pericardium may be truly constrictive and may so restrict the diastolic function of the auricles and ventricles that the filling of the latter may be so decreased as to actually interfere with the integrity of the circulation.

Anginal Failure. This term of course refers to those instances in which there are evidences of neither right nor left ventricular failure: the only symptom is angina pectoris. In one sense, it may not be strictly accurate to refer to angina pectoris as a form of heart failure. In terms of its serious significance, it certainly deserves this classification. Thinking of it as a separate form of heart failure has served perhaps in helping us to better understand this serious symptom. At any rate, the mechanism of angina pectoris has been well established in recent years. Pain results because the arteries cannot properly oxygenate the muscle of the heart under conditions of stress.

Comment: I have discussed the subject of heart failure at some length because I feel that our separating it into component parts has greatly simplified our problem and given us a much clearer understanding of the failing heart.

We have a great deal that remains to be learned, of course. We know practically nothing yet of the causes of such fundamental muscle functions as contraction, tone, etc. Before we can really understand these matters, we must learn something of the chemical, physical, and electrical processes that go on within the muscle cells. At present we must confess that we know very little about intracellular physiology. When we do come to understand these matters, I am tempted to believe that many things that we do not understand now will become clear and that our treatment of heart failure will then become much more effective and logical.

THE ETIOLOGY OF HEART DISEASE

The development that has perhaps done more than any other to give us a clearer understanding of heart disease is the realization that every case of cardiac disease has a definite cause and that only four well-known conditions, rheumatic fever, lues, hypertension, and coronary arteriosclerosis, are responsible for at least ninety per cent of all heart disease. With this has come a realization that these etiologic varieties constitute the four great types of heart disease, each completely different in its behavior from the other. We have learned a great deal about the life history, the prognosis, and the treatment of each type. Let me cite just one example to show how our emphasis on etiology has helped us.

Aortic insufficiency is seen in rheumatic disease, in luetic disease, and in arteriosclerotic disease. The murmur is the same in each. Yet, the age of onset, the duration, the course, the prognosis is completely different in these three types of hearts. Since we are no longer content simply to diagnose aortic insufficiency, but insist on knowing the type of heart in which it exists, our treatment is on an infinitely sounder and more rational basis than it was. I repeat that the emphasis on etiology is one of the greatest developments of modern cardiology.

But we really haven't yet solved the question of the cause of heart disease. We know the cause of syphilitic heart disease, but we know little concerning the cause of rheumatic fever, hypertension, or arteriosclerosis. It is discouraging that we do not know more; for it seems that we cannot go much further toward preventing rheumatic, hypertensive, or arteriosclerotic heart disease until we do. We can, however, all be heartened by the fact that the causes of these diseases are receiving as intensive study as is being devoted to any problem in medicine. I should like briefly to discuss these four types of heart disease.

Luetic Heart Disease. This variety should no longer be a medical problem. We have done our part: we have discovered its cause and we have devised a treatment which is effective if applied early. Syphilis can be and will be eradicated just so soon as the public will accept our teaching and propaganda, throw off its apathy, and take up the fight.

Rheumatic Heart Disease. We cannot expect to do very much more against this form of heart disease than we are doing at present until we know more of its cause: rheumatic fever. The latter disease is therefore our real problem. We have learned a great deal concerning this disease during the past twenty years. Some of the outstanding facts that deserve emphasis follow:

(1) Rheumatic fever is intimately but apparently indirectly associated with streptococci. The characteristic lesions of this disease are apparently produced by an allergic response on the part of hypersensitized tissues to the endotoxins of streptococci.

(2) Rheumatic fever is not an acute disease. It lasts for years. It is subject to remissions and exacerbations. It may be difficult to determine accurately whether low grade activity is present or absent. Ultimately some form of immunity develops and activity no longer manifests itself.

(3) It is a disease of childhood predominantly. Only occasionally does it develop in adults.

(4) The lesions of rheumatic fever may be widespread. The heart is nearly always involved. In addition to the heart, the peritoneum, the pleura, and the lungs are frequently affected. The joints are involved in only half of the cases. Without arthritis, the diagnosis may be difficult.

Even though rheumatic fever is a streptococcal disease, we have as yet evolved no specific treatment. The only effective measure so far discovered to lessen cardiac damage, once rheumatic fever has developed, is rest in bed for months or years. Lest we become discouraged over the fact that we have no treatment more spectacular than bed rest, let us recall that tuberculosis has been controlled chiefly by this simple measure. It accomplishes a great deal in rheumatic fever, too.

There remains, therefore, a great deal to be learned about rhemuatic fever. Once we learn more about the cause and the way in which streptococci are related to it, it does not seem unlikely that we will be able to discover some specific and effective therapy.

Hypertensive and Arteriosclerotic Heart Disease. By far the greatest number of cases of heart disease are of these etiologic types. We know clearly the course, the manifestations, and the outcome of hypertensive heart disease. We recognize that arteriosclerotic heart disease divides itself into two general types. The first is that in which cardiac pain is the predominant factor and includes acute coronary thrombosis and angina pectoris. In the second, pain is not present, or at least not outstanding. Arteriosclerotic processes narrow the lumen of the coronary arteries to a point where the nourishment of the myocardium is insufficient and the heart fails. This variety of heart failure is a very common one and for years was erroneously attributed to chronic myocarditis. We are familiar with the ar-

rhythmias and other complications of the hypertensive arteriosclerotic types of heart disease.

We, therefore, can justifiably feel that we know much about the diagnosis, the course, the mechanism, and the prognosis of hypertensive and arteriosclerotic heart disease. Where our knowledge is still very deficient lies in the fact that we know as yet so little of their fundamental causes. Without meaning to imply that their cause is necessarily the same, let us consider these two forms of heart disease together.

Our uncertainty as to the fundamental etiology of hypertension and arteriosclerosis is emphasized by the fact that so many different factors have been thought at one time or another to be their cause. Foci of infection, too much tea, coffee, nicotine, or alcohol, faulty diet, improper end products of metabolism, constipation, too much physical exertion, etc., etc. One could prolong this list ad infinitum. Most of these factors are very unsatisfactory; they do not impress us as having much to recommend them as etiologically important. But there are certain other factors that are important. These include:

(1) The fact that hypertension certainly and arteriosclerosis, less certainly, are definitely familial, a tendency toward these conditions being handed on from generation to generation.

(2) Hypertension, at least, is directly or indirectly related to certain glands of internal secretion. The hypertension that so often develops with the menopause is one suggestive example: another is the blood pressure response in certain diseases of the adrenals.

(3) Recent work has rather strongly suggested that some unknown substance elaborated by the kidneys may be a factor in producing hypertension.

(4) The stress and strain of modern life. This intangible factor is difficult for our practical minds to grasp. But there seems to be no doubt that the tension and drive necessary to carry through our aims and ambitions amid the competitive circumstances of modern life is a powerful, though poorly understood factor in producing hypertensive and arteriosclerotic heart disease.

When we are compelled to speak of the causes of hypertension and arteriosclerosis in such intangible terms, it is easy to see that our problem of preventing these diseases and their results, or of devising a rational treatment is handicapped. At present we can do nothing in regard to a kidney factor, nor can we attack the relation of the glands of internal secretion to hypertension: we do not know enough. However, we can do something regarding the other possible etiologic factors. Particularly, can we attack the familial tendency to hypertension and arteriosclerosis by having those who possess this unfortunate tendency come to us before their inheritance develops into disease. If this were done, we could remove certain contributing factors and we could often do much toward sparing such individuals from the damaging effects of the wear and tear of modern life. It would be a worthy attempt at any rate.

What can be done toward removing or lessening the stresses and strain of modern life? To some extent, we may be able to aid our patients in securing CALM, which according to Mosenthal, is the best preventive and the best treatment of hypertension. Perhaps we can teach some of our patients to emulate the Chinese, among whom hypertension was previously unknown. (I doubt if this is any longer true.) "Ah yes, the Chinese lack the spasmogenic aptitude—placid, gentle, peace loving; Buddhist—their ideal, the severe calm of Amida Buddha, with closed lids and folded hands; symbol—the lotus flower scarcely swaying over the still pool."(3) We can hardly expect to succeed in getting the modern, go-getting American to accept as an ideal the severe calm of Amida Buddha. We should be quite happy with the compromise if we can succeed in teaching them the importance and value of vacations; others, perhaps, we can help to learn the restfulness of a good hobby. I once had a patient whose hypertension, which had existed for at least four years, immediately disappeared and never returned after I persuaded him to take a three months' holiday and to take up painting as a hobby: in his opinion, the hobby saved his life (I shall not dispute this).

What else can we do? We can correct dietary and other faulty habits; we can remove foci of infection if they are definite; we can

insist on moderation in eating, drinking, smoking, and exercising. In short, let us attempt to correct every possible fault that we can discover, even though we may have no great conviction that some of our steps are logical. Until we know more of the fundamentals of these degenerative diseases, let us do more, rather than less than enough. Some day (and we hope it will be soon), we will understand these matters. Until that day comes, we must go on doing all that we can, even though this is, to some extent, empirical and probably illogical.

TREATMENT

In discussing the treatment of heart disease, so far we have said little of drugs. Without doubt, the non-medical treatment is the more important. For the main problem in treating a diseased heart is to adjust the handicapped organ to its environment; and drugs rarely play the most important role in accomplishing this. Nevertheless, drug treatment has its very important place and one of our real advances in treatment has come through a better understanding of drugs. We have developed at least one entirely new drug, quinidine, which has a limited but important place in therapeutics.

More important than this is the fact that we have learned to use digitalis much more effectively than we formerly did. We know now the approximate amount that is required to accomplish what we are after. At the same time we have learned that digitalis is not indicated in all forms of cardiac disturbances: in fact, its use may be definitely contraindicated. We have also discovered that even where its use is indicated, it can be abused; and when used improperly, may cause fatal results.

Another large advance in our therapeutic knowledge has been the realization that edema, although it is simply a symptom of a failing heart, may in itself be harmful and should be treated. The recent development of safe and effective diuretic drugs constitutes a real therapeutic advance.

SUMMARY

In spite of the serious and intensive study that it is receiving, heart disease is still one of the major medical problems. The progress that has been made and the difficulties that are still to be surmounted are discussed.

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DISCUSSION

DR. OLIN S. ALLEN (Wilmington): Mr. President, Dr. McMillan has covered the subject so thoroughly that I really do not see that there is very much—in fact, there is nothing—that I can add because I heartily agree with everything he said, in spite of the fact that I did not see his paper before its presentation and I did not talk to him about the subject before we arrived here. He certainly has covered the subject very, very well, and I appreciate it tremendously.

There are just two or three things I should like to emphasize, the same things that he went over himself, the first of which is that I stopped kidding myself long ago that I could outline a heart without the x-ray. I find that I am just not able to do it. So, with the cardiographic study, I feel sort of lost, as it were, if I do not have my x-ray picture there to see the heart size. I have been fooled on it so much that I have given it up as a bad job, and have decided, granting you that I am a very poor hand at outlining a heart, to utilize the x-ray for that purpose. So that is the very important thing that I wish to emphasize. As Dr. McMillan told you, not only the diagnosis but the prognosis of your heart case depends so much on the size of the heart.

The second thing he emphasized was that we were able with the electrocardiograph to diagnose arrhythmias. Well, that is true. I have been in the habit of, I guess, kidding myself again. You see so many of these arrhythmic fibrillators that, as they come through your office, you make a diagnosis, you get the ventricle rate, the pulse rate, and so forth and so on. In other words, maybe I have become just a little bit conceited. I put my stethoscope there and I think I can tell

a fibrillation right off or, rather, I thought I could until a couple of weeks ago, much to my surprise and chagrin. A young fellow came in; there wasn't any question from his history, so I put my stethoscope there. There wasn't any question about fibrillation. He had pulse deficit, and so forth, and so on. I thought he was pretty young for that. I hooked him up with the machine and found to my surprise that he only had auricular premature contractions. That took some of the conceit out of me when I found I had missed auricular fibrillation clinically.

That brings me back to some years ago, even before I had a cardiograph of my own, when I told Dr. McMillan "I have a boy fifteen years old who is firbrillating." He said right off the bat, "I don't believe it." "Oh, yes, he is," I said, "he is fibrillating all right. I am positive of it." This was a great many years ago. He said, "All right, bring him up, we will trace him." Much to my surprise, he had nodal rhythm. So I was wrong then, too. I didn't mind being wrong then but I certainly did object to making a mistake at the present time.

Rheumatic fever and rheumatic mitral—that seems to be one of my hobbies. I just want to say here that it really makes my heart ache to see these poor little innocent children passed over with rheumatic mitrals that can be arrested, and I would like to emphasize the fact that we ought to lay more stress on our early diagnosis of these cases, because if we ever expect to do anything in preventive medicine we must start in childhood. Then again, the outlook as far as the child is concerned depends entirely on the mother, the father, and the physicians. So that is a matter of education. All of us, I think — the family physicians—are picking these things up one hundred per cent better than they were a few years ago, but I believe there is still room for us to improve along that line.

The last thing I wish to emphasize is vacations, and, gentlemen, this applies to all physicians; don't forget that. Dr. McMillan was talking about hypertensive and arteriosclerotic disease and coronary thrombosis, and I want to say we physicians are all candidates for these. I noticed Dr. McMillan emphasized vacations two or three times. Don't forget to take vacations. I have been hammering on that, but it is difficult to get it across to physicians. You can get it across to the high pressure business man, but it is very hard to get it across to physicians; they just can't leave. But we physicians must take more vacations.

Thank you very much.

DR. JOSEPH B. WAPLES (Georgetown): Mr. President, I thoroughly enjoyed Dr. McMillan's paper, and considering that I am a general practitioner—and I might say from a small community—it has taught me much, and there is a lot more to talk to him about. We haven't a cardiograph machine; we haven't anything except the stethoscope and percussion with which to make a diagnosis, and there are a few questions I would like to ask Dr. McMillan, but before doing that I would just like to mention a case of endocarditis which it seemed to me was very interesting.

This case was that of a white man, fifty-three years old, who had had a diastolic murmur since childhood, as far as I could learn. He had had it for twenty or twenty-five years that I knew of. He was a politician. He had held various offices and had gone through much excitement without showing much trouble with his heart. He didn't give any history of ever having had any serious disease. In 1926, he had had a ruptured appendix. His heart was good through that. Last October, I was called to his house and found his heart as usual, is abdomen was distended, with quite a bit of discomfort, and in a couple of hours he started vomiting. Considering this operation that he had had a few years before, I thought probably he had a partial obstruction and rushed him to the hospital, where we were able to get an evacuation.

After he had been there for five or six weeks he ran a slight temperature sometimes, at the beginning running up as high as 102. His blood pressure ranged around 132 systolic and 78 diastolic. His blood was negative straight through. He developed chills and had a constant pain in his upper right quadrant, with some tenderness.

We had cardiograms made, and had every test that was possible. At least eight or ten doctors saw him, and I think there were eight or ten diagnoses made. He was diagnosed as

having tubercular peritonitis, abscess of the liver (because he had chills, too), chronic pancreatitis: I have forgotten all the different diagnoses that were made. He ran a very slight temperature, and died on the 4th of December.

At autopsy we found infection in practically all of his organs. His heart, on gross appearance, was of normal size. On opening the heart we found very much thickened valves, with vegetation on them, showing the cause of his death. But I don't think that many of us thought of endocarditis because his blood was negative.

All during this time he ran a leucocytosis. One day, I think, his leucocytes did get down to 6,600; the first day they were up to 12,500, and did get as high as 14,500. But his leucocyte count continually went up and down. His cardiogram was practically negative. That was of interest to me and it was interesting to the rest of the doctors, because it absolutely did not point to his heart, considering he had these valvular lesions that didn't seem to change.

I would like to ask Dr. McMillan a question. In cardiacasthma, or in asthma of any type, it is the general practitioners' habit to give them adrenalin; in cases of high blood pressure, with systolics of over 200, would it be advisable to give adrenalin? Then, too, I would like to ask, in hypertension is it right to give digitalis most of the time?

DR. ROBERT W. TOMLINSON (Wilmington): Mr. President, Dr. McMillan, and Members of the Society: I count it a rare and inestimable privilege to be able to say something after so instructive a paper, so charmingly proferred.

It was my good fortune some six years ago to wait upon Dr. McMillan at his office and solicit the privilege of being in attendance at his clinic at the Philadelphia General Hospital. In the course of our conversation, noticing the softness and the music of his enunciation, I felt that such could only be indicative of his emanation from the Southland, and I was agreeably surprised to learn that he came from Mobile.

He had already assured me of his willingness to have me come as a student and when I acquainted him with the fact that my wife's brother-in-law was a doctor in Mobile he very sweetly said, "I told you just now that we would welcome you at the Hospital and now I will add that we will do it with open arms" —a kindly verbal gesture which encouraged an aspiring, embarrassed man.

Philadelphia has had many men of note enrolled on its scroll of honor for professional achievement, and I think that now it is going to have to emboss on such parchment the name of Dr. Thomas McMillan—modest in demeanor, ultraconservative, possessed of an inestimable degree of knowledge pertinent to cardiac dyscrasia, it was always an extreme privilege and happiness to be able to bask in the sunshine of his illuminating instruction. It would be worth the while of every man here to frequent that clinic where he will see a beehive of industry and of research conducted in an orderly and most efficient manner.

I have seen Dr. McMillan stand and listen to the expressed opinions of his assistants when, at the time, I felt that he held a conclusion different from theirs, and yet with ineffable sweetness I would see him correct them and instruct them, never making a man feel small, but enhancing the acumen which he could evince in solving difficult clinical problems at the bedside.

I am not going to tire you with further eulogistic phrases. I simply want to express my thanks to Dr. McMillan for another opportunity to gain information, in which I am sure you will all join me.

DR. EDGAR R. MILLER (Wilmington): I do not wish to to lose an opportunity to express my appreciation for the privilege of hearing Dr. McMillan's paper. He has well covered the subject. However, from a general practitioner's point of view, as Dr. Waples has said, I would like to say that there are two types of patients who come into our offices in regard to heart disease. One is the type who thinks he has heart disease, and does not. The other type has has heart disease but does not think he has.

We must remember as physicians that many times, in examining a patient by the routine methods of examination—taking his history, the physical examination, particularly with the stethoscope—the heart may appear

to us to be perfectly normal, and yet—this concerns particularly the arrhythmias, those that are paroxysmal in type, transient in nature—probably if we had seen that patient twelve hours before, say at three o'clock in the morning, our opinion would not have been the same.

We may say to the patient, "Why, your heart is perfectly all right," but I think we fail to realize that sometimes after a food debauch of the previous night, or excessive smoking, or many other factors ranging anywhere from ptosis of the stomach to neurogenic influences such as excitement or some family squall, the heart can cut capers which are quite disturbing to the patient and probably would be disturbing to us if we could listen to the heart at that time.

In other words, I have had many patients come in saying, "Why, my heart was said by Dr. So-and-so to be perfectly normal." Electrocardiograms have been taken and x-rays have been taken and everything has been normal, but such things as premature beats, whether auricular or ventricular contractions, or paroxysmal tachycardias do not always present themselves at the time of the examination. So I think that group of patients that come to the physician make up a very large per cent who really do have cardiac disturbances which at the time of the examination, even to a cardiologist, may pass as perfectly normal.

Some of these patients can be cured by cutting down on their smoking, or giving them a ptosis belt, or altering their diet.

So, as I say, as internists or general practitioners, let us not forget the fact that many people are disturbed from the cardiac point of view, and justly so, and yet upon examination by every available means they may appear normal.

Then the second group of patients which I would like to say a word about is the cardiac patient who is over-treated. Just as it takes more than one swallow to make a summer, it takes more than a murmur to make heart disease. As Dr. McMillan said and as Dr. White has said, most systolic murmurs are not cardiac disease.

I know in examining nurses as candidates for admission to the hospitals, I have been impressed by the great number of murmurs that one hears, and digitalis is often prescribed because of an innocent systolic murmur. A patient is often put to bed for periods of weeks because of a systolic murmur.

It seems to me that before prescribing any definite treatment and making a patient heart-conscious or condemning a patient as a cardiac invalid, definite investigation should be made and the diagnosis should be verified. Before we put a patient to bed unjustly, sometimes because of lesions which are not at all harmful, we should be perfectly sure of ourselves.

As an example of that point, Dr. White was telling a story about how he went to Roanoke, Virginia, to give a talk on heart disease. A physician who was at the meeting told him that he had a boy with a serious cardiac ailment whom he had kept in bed for a period of several months, and he would like him to see this case in consultation. So Dr. White went to the home and examined the patient. It turned out that the boy did have a very loud systolic blow, but it was an inter-ventricular septal defect, perfectly harmless, which the boy had probably had all his life, and which would even warrant his carrying on the normal activities of life. The result was that the boy was allowed to get up and go back to school, and he got along perfectly well.

So, as I say, Dr. McMillan's talk did cover things very thoroughly, but from our point of view as internists and general practitioners we must keep in mind the cases that have cardiac disturbances, yet are difficult to ascertain even from the cardiologist's point of view; and second, that group of cases that have some murmurs and yet are not definite cardiac disease.

DR. MCMILLAN: I believe the only specific question that I was asked was with regard to asthma and the use of adrenalin, particularly if there is a high blood pressure. I do not like to give adrenalin ever to a patient with heart disease, particularly if there is high blood pressure. My reason for this is because I have seen what a dog's heart does when you give it adrenalin. Adrenalin not only causes the heart to beat rapidly, but it causes it to do

what is referred to physiologically as hypercontract. It contracts much more vigorously than it does without adrenalin. If you give a dog a good big dose of adrenalin and then look at that dog's heart you will find the whole surface of the heart studded with little minute hemorrhages because the ventricle has beaten so hard that it has caused little surface capillaries to break. For this reason I do not like to give adrenalin to a person with high blood pressure unless I am very certain that the asthma is not cardiac in type.

As to the use of digitalis in high blood pressure, as far as I know, there is no contraindication. In fact, a great many people feel that it is indicated. They feel that digitalis has a tendency to stabilize blood pressure. They feel if it is low digitalis tends to make it approach the normal; if it is high, it tends to make it come down to the level it should occupy.

Dr. Allen touched on the early diagnosis of mitral stenosis. I should like to say it is most important to know when our cases have early rheumatic hearts because it is then that we can do something in the way of effective treatment.

The proper way to diagnose early rheumatic lesions is to recognize rheumatic fever in its atypical manifestations. That isn't easy to do. Diagnosis of rheumatic fever is not easy. I only have to say this to emphasize this fact: If you take 100 cases of mitral stenosis and go over them and ask those patients, "Did you ever have rheumatic fever," fifty out of the hundred will tell you that they never had rheumatic fever. Yet they must have had rheumatic fever or they wouldn't have this rheumatic mitral stenosis. The answer is, they had rheumatic fever but it existed in some atypical form and was not recognized by their physicians as rheumatic fever, and no diagnosis of this condition was ever made. So I think as general practitioners we should school ourselves and should have before us the possibility that rheumatic fever may be a most atypical disease. When we hear a systolic murmur develop in the course of such atypical disease then we should be quite suspicious of some cardiac lesion.

I am indebted to the other gentlemen for their discussion of this paper and express my gratitude to all of you.

THE THERAPEUTICS OF IRON AND COPPER

EDWARD PODOLSKY, M. D.
Brooklyn, N. Y.

One of the most useful and one of the oldest in length of service to man therapeutically is iron. When iron was first used the reasons for its use were based on mysticism and empiricism; no scientific explanations were attempted for its indications. It was used in its crudest form. The person probably to have prescribed iron first was Melampus, the ancient Greek physician, whom his countrymen believed to have been the first mortal to have practiced the healing art. The form of iron he prescribed was rust. Later physicians began to prescribe iron in the form of drinking water in which the "glowing iron is quenched in the smithies," a crude method but probably effective after a fashion, for it persisted for quite a length of time.

The earliest scientific evidence for the use of iron was the discovery by Menghinis in 1846 that iron was a normal and characteristic constituent of the blood. Menghinis also found that a diet rich in iron caused an increase in the iron content of the blood. In 1832 Foedisch found that patients suffering from what was then a rather common ailment, chlorosis, had a much lower content of iron in their blood than normal persons. Ten years later Andral, Gavarets and Delafond found that when iron was fed to these patients there was an increase in the number of red blood cells and a perceptible improvement in the patient's condition.

After these scientific contributions to the question there was much controversy, particularly during the years from 1890 to 1905 as to the absorption and utilization of different forms of iron, centering chiefly of food iron, complex organic compounds, and inorganic iron salts.

With regard to food iron there can be no question as to absorption and utilization.

During the early growth period of man and animals in which there is rapid development and extension of the various tissues, the hemoglobin increases satisfactorily on an adequate diet. Among the foods rich in iron, spinach, apricots, peaches and prunes are the best known.

As regards inorganic iron, the experimental work of such men like MaCollum, Hall, Quincke and Gaule showed, by microscopic examination of the intestinal tract that inorganic iron salts were absorbed mainly in the upper portions of the small intestines. Gaule also demonstrated the absorption of iron by administering an 0.06 per cent solution of ferric chloride by stomach tube and proving by chemical tests the presence of iron in the lymph of the thoracic duct three-quarters of an hour later. Additional evidence of the absorption of inorganic iron was also obtained by Hoffmann and Rabe who used both man and animals as experimental subjects, and made quantitative comparative studies of the iron ingested and excreted.

Kunkel, Cloetaa and Aberhalden proved by experimental studies on animals that inorganic iron salts were absorbed, stored and subsequently utilized by the body for the formation of hemoglobin. The work of Tartakowsky confirmed the above findings and indicated that in addition to their utilization for the formation of hemoglobin, inorganic iron salts had a pronounced stimulating effect upon the blood-forming organs, especially when the hemoglobin was at a low level, as in chlorosis.

At the time when it was believed that the inorganic iron salts were not absorbed, various organic iron compounds were extensively used. With the definite knowledge that inorganic iron salts are readily absorbed and utilized this form of iron is being used quite extensively.

Since approximately 80 per cent of the total iron content of the body is contained in the hemoglobin, it is evident that during the growth period there is a great demand for iron to meet the rapid increase in hemoglobin formation. The usual food of man is not particularly rich in iron. Sherman in 1907 made an extensive study of iron metabolism and found that the average intake of iron was 11 to 19 mgs. In deficient diets or where the appetite is poor the iron intake may fall as low as 4 mgs. The quantity of iron excreted in the urine and feces is almost equal to the intake. Studies made on two professional fasting subjects, showed a daily excretion in the feces of 7 and 8 mgs., respectively. It is therefore quite evident that under the most favorable dietary conditions the amount of iron derived from food is barely sufficient to cover our physiological demands, i. e., to provide for catabolic losses, and does not allow any excess for iron. It would appear that this element has in large amounts a decidedly stimulating effect upon the blood-forming organs and upon metabolic processes in other tissues.

Many different forms of iron have been used orally and hyperdermically for many years in the treatment of anemias. As a general rule the results have not been very satisfactory. Recent clinical work, however, definitely shows that previous failures in the treatment of many of the cases of secondary anemia have been due in a large measure to inadequate doses of iron administered. In 1926 Goodall advocated the use of large doses of iron in the treatment of anemias. He regarded the massive doses of iron not so much a supply of iron but more as a stimulus to the utilization of iron, i. e., to the formation of certain substances necessary for the production of hemoglobin.

There are various forms of iron for oral and intravenous administration. Among the most commonly used are ferric oxide, ferrous carbonate, and reduced iron. These require an acid medium for absorption, which is furnished by the free hydrochloric acid normally present in the stomach.

The disease in which iron is most often indicated, of course, is anemia. As is well known in pernicious anemia there is an absence of free hydrochloric acid in the stomach. Consequently, the greater part of the water soluble iron compounds are not absorbed. This point is emphasized by the negative results of Hart, Elvehjem, Wadell and Herrin using ordinary iron oxide, and by Doan, Sabin and Forkner using magnetic ferric oxide produced by Baudisch. Likewise, the experimental work of Mitchell and Schmidt indicates that

the more soluble the iron salt and therefore the more available for absorption, the more pronounced is its therapeutic effect.

Many iron compounds such as ferric chloride and ferric sulphide exhibit a marked astringent effect upon the gastro-intestinal mucosa, and if repeated for long period, strong astringents tend to impair gastric secretion and retard absorption. Of the iron compounds, perhaps the most desirable from the therapeutic point of view is ferric ammonium citrate, which possesses to a minimum any of the undesirable effects of iron compounds.

THE THERAPEUTICS OF COPPER

Copper, within recent years, has been found to be very intimately associated with iron metabolism. Stone found that a deficiency in copper interferes with the assimilation of iron and is apparently an important factor in the etiology of certain infantile and adult anemias. McHargue, Healy and Hill ascertained that copper has an important function in the formation of hemoglobin. The feeding of copper and iron combined at times may have an unusually favorable effect or again may not exceed the influence of iron alone.

The experiments or Flinn and Inouge indicate that copper exerts an influence on the hematopoietic system and on the metabolism of the body as a whole. They administered and determined the distribution of copper in the urine, feces and viscera. The greater part of the stored copper was confined to the liver, as in human livers obtained at autopsy. Very little copper was deposited in the bone, nor did it replace calcium. No methemoglobin formation followed prolonged copper ingestion, nor was there any evidence of hemolytic effect.

Evvard, Nelson and Sewell found that copper is of considerable nutritive importance. Rats and swine made better gains and exhibited higher food utilization when copper was incorporated in the rations. The greater part of the copper was confined to the liver. It is possible that the medicinal and nutritive value of liver and its proper functioning may be somehow related to this metal.

Dwyer found that copper is of some value in the treatment of anemia in selected types of cases, particularly where there is a nutritional factor. Lewis found that hemoglobin regeneration and response of the red blood cell was definite with iron alone, but not so definite as when copper supplemented the iron. In general it has been found that iron and copper given in combination to children with nutritional and secondary anemia was more effective than iron given alone.

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The American Association of Obstetricians, Gynecologists and Abdominal Surgeons announces that the annual foundation prize for this year will be $100. Those eligible include only (1) interns, residents, or graduate students in Obstetrics, Gynecology and Abdominal Surgery, and (2) physicians (M. D. degree) who are actually practicing or teaching Obstetrics, Gynecology or Abdominal Surgery.

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For further details, address Dr. James R. Bloss, secretary, 418 11th street, Huntington, W. Va.

EDITORIAL

DELAWARE STATE MEDICAL JOURNAL

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VOL. XI MARCH, 1939 No. 3

WANTED—BIOGRAPHIES AND PHOTOGRAPHS

The year 1939 marks the Sesqui-Centennial of the Medical Society of Delaware, which was incorporated by special act of the legislature on February 3, 1789, thus making it the third oldest medical society and the second oldest medical corporation in the United States. New Jersey holds first place in both categories, with Massachusetts in second place as a society.

Naturally, when a society reaches the ancient and honorable status of one hundred and fifty years, something bigger and better has to be done about it than the ordinary anniversary. Ambitious plans for the celebration, to be held in Wilmington next October, are being discussed, and we doubt not that the powers that be will see to it that we shall have something to remember for a long time.

In connection with our long history we have an earnest appeal to make to our members and friends. We need certain historical and biographical data to make our records complete, and we urge upon all of you to send to the Medical Society of Delaware—in care of the Delaware Academy of Medicine, Wilmington—all biographical data and photographs to which you have access and which relate to physicians of Delaware, especially those prior to 1875. We guarantee safekeeping of this material under lock and key, and its safe return after it has been copied, abstracted or photostated. Especially desired is the biography and photograph of every physician who has served as president of the Society, which, if successful, will make our collection almost unique in this country. If all you can send is a mere reference to some little known book, please do your bit. Or if it be an oil painting of one of our earlier physicians, send us a photographic print of it.

Raking up old material such as is desired may be a tedious and a dusty enterprise, but the results will amply repay you. In advance, we thank you for your interest and your help.

In addition to the biographies and photographs, if any data concerning medical meetings or procedures are available, kindly lend them to us.

This sort of endeavor is well worth while, and will increase in value as time passes by.

THE CONCEIT OF THE CENTURY

Place: A local hospital.

Time: Recently.

Dr. A. (to Dr. B.): "You know, I've often wondered what's going to happen to this place when I pass on!"

Delaware Academy of Medicine

Officers and directors to serve for the year were elected at the annual meeting of the Academy, January 24, 1939.

Dr. Lewis B. Flinn was re-elected president; other officers are: First vice president, Dr. Willard F. Preston; second vice president, Dr. John C. Pierson; secretary, Dr. John H. Mullin; treasurer, Dr. W. H. Kraemer; chairman of the library committee, Dr. I. M. Flinn, Jr.; chairman of the scientific committee, Dr. O. S. Allen; chairman of the admission committee, Dr. J. D. Niles; representative of the Medical Society of Delaware, Dr. W. O. LaMotte; representatives of the Homeopathic Medical Society, Dr. V. D. Washburn; and representative of the Delaware State Dental Society, Dr. E. E. Veasey.

Two new members were elected to the board of directors: Mrs. Ernest du Pont and Mr. W. P. Allen; those now serving on the board are: Dr. C. M. A. Stine and Messrs. Walter S. Carpenter, H. F. du Pont, A. L. Bailey, and S. D. Townsend. The new officers, directors and committees will assume office on April 1, 1939.

Newly-elected members of the various committees are: Dr. G. H. Gehrmann, library committee; Dr. C. L. Hudiburg, admission committee; Dr. Charles Levy, scientific committee; and Drs. E. R. Miller and J. Draper Brown, executive committee.

The following were elected to membership in the Academy: Dr. Norman L. Cutler, Medical Arts Building; Dr. John H. Foulger, Haskell Laboratory; Dr. Edmund G. Laird, 1208 Delaware avenue; Dr. John W. Maroney, 1100 Broom street; Dr. E. T. O'Donnell, 1004 Jefferson street; Dr. Alfred R. Shands, Delaware Trust Building; Dr. J. A. Shapiro, Citizens Bank Building, and Dr. Millard F. Squires, Jr., Richardson Park.

The annual report made by the president noted the steady progress made by the Academy since its founding in 1930. It is significant that the small group of physicians and dentists instrumental in its establishment has increased in number until it is now well over a hundred. The growth of the organization during 1938, with the increased use of both the reading room and the auditorium by members and allied educational institutions, as stated in the president's report, indicates that the Academy is fulfilling a useful purpose in the community, and has come to be looked upon as the medical center of the state.

The Academy, through its scientific committee, attempts to arrange meetings somewhat different in type from those available through the state and county society or hospital staff programs, and since its founding nearly ten years ago, has arranged annually several lectures and symposia presented by well-known authorities among the medical and dental professions. This is in keeping with the general policy of the Academy—to offer something of an educational, scientific nature which complements the meetings and exhibits held by the other medical organizations.

MISCELLANEOUS

Van Meter Prize Award

The American Association for the Study of Goiter again offers the Van Meter prize award of three hundred dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The award will be made at the annual meeting of the Association which will be held in Cincinnati, Ohio, on May 22, 23 and 24, 1939, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length, must be presented in English; and a typewritten double-spaced copy sent to the corresponding secretary, Dr. W. Blair Mosser, 133 Biddle street, Kane, Pa., not later than April 15, 1939. The committee, who will review the manuscripts, is composed of men well qualified to judge the merits of the competing essays.

A place will be reserved on the program of the annual meeting for presentation of the prize award essay by the author if it is possible for him to attend. The essay will be published in the annual proceedings of the Association. This will not prevent its further publication, however, in any Journal selected by the author.

1789—MEDICAL SOCIETY OF DELAWARE—1939

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Delegate to A. D. A.: P. A. Traynor, Wilmington. *Alternate*: J. A. Bounds, Seaford.

Washington Assembly

"The eyes of the medical world are on Washington," according to an announcement just received from the Medical Society of the District of Columbia which states that the Annual Scientific Assembly will be held April 25, 26 and 27, 1939, in the Mayflower Hotel in Washington.

Gastroenterology in all its phases will be the subject covered in the three-day postgraduate course, offering about fifty-two papers, panels and round tables on the subject. The program lists, in addition to many prominent physicians of the faculties in Washington, the following from elsewhere: Dr. Lewellys L. Barker, Baltimore; Dr. Fred Rankin, Lexington, Kentucky; Dr. L. M. Hurxthal, Boston; Dr. R. J. Coffey, formerly of Mayo Clinic; Dr. B. B. Vincent Lyon, Philadelphia; Dr. E. H. Gaither, Baltimore; Dr. P. P. Vinson, Richmond; Dr. Eloise B. Cram, National Health Institute.

Luncheons, stag meeting, banquet, and entertainment for visiting wives are provided by the Society.

Reservations are being taken by Theodore Wiprud, secretary of the Society, 1718 M street, N. W., who will forward full information on request.

BOOK REVIEW

Surgical Treatment of Hand and Forearm Infections. By A. C. J. Brickel, M. D., Department of Anatomy and Surgery, Western Reserve University. Pp. 300, with 191 illustrations and 10 color plates. Cloth. Price, $7.50. St. Louis: C. V. Mosby Company, 1939.

This volume represents the researches of many years, in a field where the results are not always satisfactory and occasionally crippling. The book abounds with new pointers on the anatomy and the surgery of this field, which are made unusually plain by the numerous excellent illustrations. These alone are worth the price of the book. Special chapters are devoted to the general principles of hand infections, the routes by which they spread, human bites, anesthesia, physiotherapy and medicolegal considerations.

This book is the logical successor to that of the late Kanavel, and should be part of the armamentarium of all surgeons who operate on such infections, especially industrial surgeons.